

TECHNICAL MEMORANDUM NO 10

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HUMAN HEALTH RISK ASSESSMENT 903 PAD, MOUND, AND EAST TRENCHES AREAS OPERABLE UNIT NO 2 TOXICITY ASSESSMENT

DRAFT

ROCKY FLATS PLANT

U S DEPARTMENT OF ENERGY Rocky Flats Plant Golden, Colorado

ENVIRONMENTAL MANAGEMENT DEPARTMENT

July 1993

DOCUMENT CLASSIFICATION REVIEW WAIVER PER CLASSIFICATION OFFICE

Woodward-Clyde Federal Services

Engineering & sciences applied to the earth & its environment

July 22, 1993

Ms Annette Primrose EG&G Rocky Flats, Inc PO Box 454, Building 080 Golden, CO 80402

RE Submittal of Draft Technical Memorandum No 10
Toxicity Assessment
Rocky Flats Plant, Operable Unit No 2
BOA BA 56801 PB, Requisition BA 71956 PB

Dear Annette

Enclosed are 12 copies of the Draft Technical Memorandum No 10, Toxicity Assessment, 903 Pad, Mound, and East Trenches Area, Operable Unit 2 for your review and comment

We look forward to receiving your comments

Cathlan M Cowe

Sincerely,

Kathleen M Power OU-2 Project Manager

Y Which hell Patricia Westphal

Risk Assessment Task Manager

TECHNICAL MEMORANDUM NO. 10

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TABLE OF CONTENTS

1 0	INTI	RODUCTION	1-1
2 0	ВАС	KGROUND	2-1
	2 1	NONCARCINOGENIC TOXICITY FACTORS FOR CHEMICALS	2-1
	22	CARCINOGENIC TOXICITY FACTORS FOR CHEMICALS	2-2
	23	RADIATION TOXICITY FACTORS	2-5
3 0	тох	ICITY FACTORS	3-1
	3 1	INHALATION AND ORAL TOXICITY FACTORS	3-1
	32	DERMAL TOXICITY FACTORS	3-1
	33	DERMAL PERMEABILITY CONSTANTS AND ABSORPTION	
		FACTORS .	3-5
10	REF	ERENCES	4-1
		The state of the s	



The purpose of a toxicity assessment is to evaluate the toxicity and estimate the dose-response relationship for chemicals of concern. This Toxicity Assessment Technical Memorandum No 10 contains the carcinogenic and noncarcinogenic toxicity factors which are proposed for use in the human health risk assessment to describe the toxicity of potential chemicals of concern at Operable Unit No 2 at the Rocky Flats Plant in Golden, Colorado. Potential chemicals of concern are chemicals and radionuclides whose presence at OU-2 is related to plant operations. In the OU-2 human health risk assessment, concentrations of selected chemicals of concern will be multiplied by chemical intakes to estimate dose, and those doses will be combined with the toxicity factors presented in this technical memorandum to estimate potential excess lifetime cancer risk and noncarcinogenic health hazards.

Chemicals of concern for OU-2 were selected using established procedures from EPA guidance (USEPA 1989) The details and the results of the selection process are presented in Technical Memorandum No 9, Selection of Chemicals of Concern (USDOE July 1993)

Noncarcinogenic responses are typically characterized by a threshold value. The threshold value is a dose of a chemical below which adverse effects are not expected to occur. Above the threshold dose, protective physiological mechanisms may not be effective. EPA policy is to assume that carcinogenic responses have no threshold. This assumption results in some finite cancer risk at any dose, no matter how small (USEPA 1989)

The two principal indexes of toxicity for chemicals (nonradioactive) are the reference dose (RfD) and slope factor (SF). These values are derived by the EPA for the most commonly occurring and the most toxic chemicals generally associated with chemical releases to the environment (USEPA 1992a and 1993). An RfD can be considered a threshold dose that incorporates a number of safety factors to ensure that it is protective of the health of all human populations, including sensitive subgroups (e.g., children and the elderly). RfDs are typically reported as dose of a chemical per unit body weight per day. As long as the chronic daily intake of a chemical is less than the chronic reference dose, noncarcinogenic health effects are not expected to occur.

Slope factors are used to estimate the upper-bound probability of an individual developing cancer as a result of exposure to a potential carcinogen Potential carcinogens according to



EPA are given an EPA weight-of-evidence classification. The weight of evidence system is used as a means to describe the level of confidence in the data used to identify a chemical as a human carcinogen (USEPA 1989)

A toxicity evaluation of radionuclides has certain fundamental differences from nonradioactive chemicals. Adverse effects of internal exposure to radionuclides are related to the energy level and residence time in the body of radionuclides deposited in various body tissues. Duration of exposure is determined by the residence time of the radionuclide. Adverse health effects of external exposure to radionuclides are determined by the energy level and duration of the exposure (i.e., time spent at the exposure point)

EPA assumes that any dose of radiation has the potential to produce carcinogenic effects (no threshold) EPA does not recommend the evaluation of noncarcinogenic effects of radionuclides because the impacts have been shown to be insignificant compared to carcinogenic effects at most EPA Superfund sites with radionuclide contamination (USEPA 1989). The relationship between dose and carcinogenicity is relatively well described for high doses of most types of radiation (i.e., alpha and beta particles and gamma rays) (Eisenbud 1987). Exposure to multiple radionuclides is often expressed in terms of total radiation dose by consideration of the target organ effects of individual radionuclides. The total radiation dose to the human body is of greater concern in a toxicity assessment than the individual contributions of radiation from radionuclides.

USEPA has developed both internal (i.e., inhalation and ingestion) and external slope factors for the carcinogenic response to radionuclide exposure (USEPA 1992a and 1993). Although more recent data on radionuclide dose-response relationships than that used to develop the EPA slope factors is available (i.e., the BEIR V report and ICRP Publication No 60), it has not yet been approved by EPA. Therefore, the currently approved EPA slope factors (USEPA 1992a) will be used in the toxicity assessment section of the human health risk assessment for OU-2.

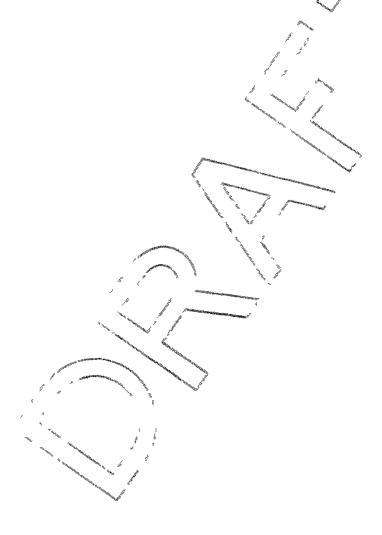
The RfD and SF values which will be used in the OU-2 risk assessment were obtained from the following sources

- EPA's Integrated Risk Information System on-line database (USEPA 1993)
- EPA's Health Effects Assessment Summary Tables (USEPA 1992a)



1-2

Section 20 of this toxicity assessment technical memorandum discusses the basis of toxicity factors. Noncarcinogenic toxicity factors are addressed in Section 21, carcinogenic factors in Section 22, and radiation factors in Section 23. Section 30 presents the chemical-specific toxicity factors which will be used in the risk assessment to estimate toxicity for the chemicals of concern in groundwater and in surface and subsurface soils at OU-2. Section 31 presents toxicity factors for inhalation and ingestion exposures. Section 32 discusses dermal exposure, including the use of oral toxicity factors for dermal exposure, and presents the chemical-specific dermal permeability constants which will be used in the OU-2 human health risk assessment. Section 40 provides the references used in this technical memorandum.





The following sections discuss the basis for each of the three types of toxicity factors proposed for use in the toxicity assessment of the OU-2 human health risk assessment. The three types of toxicity factors represent noncarcinogenic health effects from exposure to chemicals, carcinogenic health effects from exposure to radionuclides

2.1 NONCARCINOGENIC TOXICITY FACTORS FOR CHEMICALS

Substances that produce noncarcinogenic effects are generally thought to have a threshold dose below which there are no observable adverse health effects. In developing a toxicity value for noncarcinogenic effects, the approach used by EPA is to identify this threshold dose, or no-observed-adverse-effect level (NOAEL), through studies with experimental animals or from epidemiological (human) studies. A NOAEL is defined as an experimentally (or epidemiologically) determined highest dose at which there was no statistically or biologically significant effect of concern. For certain substances, only a LOAEL, or lowest-observed-adverse-effect level, has been determined. This is the lowest dose of a substance that produces either a statistically or biologically significant indication of the critical toxic effect. The NOAEL or the LOAEL may be used in conjunction with appropriate uncertainty factors to calculate the RfD (reference dose) of a particular chemical (USEPA 1989)

The majority of our toxicological knowledge of chemicals comes from experiments on laboratory animals. Experimental animal data historically have been relied upon by regulatory agencies and other expert groups to assess the hazards of human chemical exposures. Although this reliance has been generally supported by empirical observation, there are known interspecies differences in chemical absorption, metabolism, excretion, and toxic responses. There are also uncertainties concerning the relevance of animal studies using exposure routes (i.e., intravenous injection) that differ from the human exposure routes under consideration. Additionally, the extrapolation of results from short-term or subchronic animal studies to long-term exposures in human has inherent uncertainty (USEPA 1989)

Despite the limitations of experimental animal data, such information is essential for chemical toxicity assessment, especially in the absence of human epidemiological evidence. The uncertainty factors used in the derivation of RfDs are intended to compensate for data



limitations The use of uncertainty factors is conservative by design and is meant to result in protective RfD values (USEPA 1989)

The EPA has developed various types of RfDs depending on the exposure route (ingestion or inhalation), the critical effect, and the length of exposure being evaluated (chronic or subchronic). The EPA bases the RfD on the most sensitive animal species tested (i.e., the species that experiences adverse effects at the lowest dose). RfDs are typically calculated by dividing the NOAEL (or LOAEL) by uncertainty factors, which generally range from 10 to 1000 EPA has developed a standard set of uncertainty factors to account for variations in the sensitivity of individuals within a population and the extrapolation of data from experimental animals to humans. The RfD is expressed in units of milligrams of chemical per kilogram of body weight per day (mg/kg-day) for oral exposure. Reference air concentrations (RfCs) expressed in milligrams of chemical per cubic meter of air (mg/m³) may be available to evaluate inhalation exposure. A body weight of 70 kg and a respiration rate of 20 m³/day are used to convert the RfC to a dose (mg/kg-day). The methodology for deriving RfDs is more fully described in the EPA's current human health risk assessment guidance (USEPA 1989).

The EPA defines a chronic RfD as an estimate of a daily exposure level for the human population that is unlikely to result in deleterious effects during a lifetime (70 years, according to EPA guidance). A chronic RfD is used to evaluate the potential noncarcinogenic hazards associated with long-term chemical exposures (7 years to a lifetime). Subchronic RfDs have been developed to characterize potential noncarcinogenic hazards associated with short-term chemical exposures. The EPA defines subchronic exposure as periods ranging from 2 weeks to 7 years (USEPA 1989). Subchronic RfDs tend to be higher for many chemicals, generally by one order of magnitude, than chronic RfDs because of the shorter exposure duration

Section 30 presents the gral and inhalation RfDs for each chemical of concern for OU-2

2.2 CARCINOGENIC/TOXICITY FACTORS FOR CHEMICALS

In estimating the risk posed by potential carcinogens, it is the common practice of the EPA to conservatively assume that any exposure level is associated with a finite probability, however minute, of producing a carcinogenic response EPA assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation. This mechanism for carcinogenicity is referred to as "non-threshold" since there is theoretically no level of exposure for such a substance that does not pose a small, though finite, probability of producing a carcinogenic response. The EPA uses an evaluation process in which the



substance is assigned a weight-of-evidence classification. The weight-of-evidence classification describes the degree of confidence likelihood, based on scientific evidence, that the substance is a human carcinogen. Table 2-1 defines the current EPA weight-of-evidence classification system. A slope factor (SF) is then calculated that quantitatively defines the relationship between average lifetime dose and carcinogenic risk (USEPA 1989).

Slope factors for most chemicals are usually based upon the results of animal studies. The majority of our toxicological knowledge of chemicals comes from experiments on laboratory animals. Experimental animal data historically have been relied upon by regulatory agencies and other expert groups to assess the hazards of human chemical exposures. Although this reliance has been generally supported by empirical observation, there are known interspecies differences in chemical absorption, metabolism, excretion, and toxic responses. There are also uncertainties concerning the relevance of animal studies using exposure routes (i.e., intravenous injection) that differ from the human exposure routes under consideration (USEPA 1989)

Despite the limitations of experimental animal data, such information is essential for chemical toxicity assessment, especially in the absence of human epidemiological evidence. There is uncertainty whether all animal carcinogens are also carcinogenic in humans. While many chemical substances are carcinogenic in one or more animal species, only a small number of chemical substances are known to be human carcinogens. The EPA assumes that humans are as sensitive to all animal carcinogens as the most sensitive animal species. This policy decision is designed to prevent underestimating risk and introduces the potential to overestimate carcinogenic risk (USEPA 1989).

A number of mathematical models and procedures have been developed to extrapolate from carcinogenic responses observed at high doses in experimental animals to responses expected at low doses in humans. The EPA uses a conservative mathematical model, the linearized multistage model, for low-dose extrapolation. The EPA further conservatively estimates the upper 95th percentile confidence limit of the slope of the resulting dose-response curve. This SF value, expressed in units of risk per mg/kg-day or (mg/kg-day)¹, is used to convert the average daily intake of chemical, averaged over a lifetime, to an excess incremental lifetime cancer risk. This represents an estimation of an upper-bound incremental lifetime probability that an individual will develop cancer as a result of exposure to a potential carcinogen. This model provides a conservative estimate of cancer risk at low doses, and is likely to overestimate the actual cancer risk. The EPA acknowledges that actual slope factors are likely to be between zero and the estimate provided by the linearized multistage model (USEPA 1989)



TABLE 2-1 EPA CARCINOGENICITY WEIGHT-OF-EVIDENCE CLASSIFICATIONS

Group A	Human carcinogen (sufficient evidence of carcinogenciity in humans)
Group B	Probable human carcinogen
Group B1	Limited evidence of carcinogenicity in humans
Group B2	Sufficient evidence of carcionogenicity in animals and inadequate or lack of evidence in humans
Group C	Possible human carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)
Group D	Not classifiable as a human carcinogen (inadequate or no evidence)
Group E	Evidence of noncarcinogen for humans (no evidence of carcinogenicity in adequate studies)



The slope factors and weight-of-evidence classification for the chemicals of concern at OU-2 are presented in Section 3.0

2.3 RADIATION TOXICITY FACTORS

EPA provides guidance in the Health Effects Assessment Summary Tables (USEPA 1992) that lists cancer slope factors for selected radionuclides of potential concern at Superfund sites. These values were calculated by the Office of Radiation Programs and are intended for use in human health risk assessments. EPA classifies all radionuclides as Group A carcinogens based on the extensive weight-of-evidence provided by epidemiological studies of radiation-induced cancers in humans. According to EPA, potential health risks at most CERCLA radiation sites are usually based on the radiotoxicity, rather than chemical toxicity.

Radionuclides that enter the body may become systemically incorporated and emit alpha, beta or gamma radiation for the duration of the radionuclide's lifetime. The potential adverse effects of radiation are proportional to energy deposition. The energy deposited in tissues is proportional to the decay rate and the type of radiation of a radionuclide, and not its mass (USEPA 1989). Radionuclide intake is typically expressed in terms of activity, either Curies or Becquerels (Bqs) rather than mass (mg). Activity refers to the number of nuclear disintegrations per unit time. The historic unit of activity is the Curie (Ci) that is equal to 3.7 x 10¹⁰ disintegrations per second. The SI (Systeme Internationale) unit of activity is the Bq, equal to one disintegration per second. (1 Bq = 2.7 x 10¹¹ Ci). USEPA slope factors are provided in both units, picocurie (pCi) (pCi or 1 x 10¹² Ci) and Bq. This technical memorandum uses the historic unit of activity, the Curie

USEPA slope factors for radionuclides are characterized as best estimates (median or 50th percentile) of the age averaged, lifetime excess total cancer incidence (fatal and nonfatal) risk per unit exposure to a radionuclide. The USEPA slope factors are based on the unique chemical, metabolic and radiological properties of individual radionuclides. They were calculated using a non-threshold, linear dose-response model. The model accounts for the amount of radionuclide absorbed into the body, distribution and retention, as well as the age, sex and weight of an average individual. Therefore, USEPA slope factors for radionuclides are not expressed as a function of body weight or time, and do not require corrections for absorption or lung transfer efficiencies (USEPA 1992)

Ingestion and inhalation slope factors estimate risk per unit of activity inhaled or ingested expressed as risk/pCi External exposure slope factors are best estimates of risk for each year



of exposure to external radiation from photon-emitting radionuclides distributed uniformly in a thick layer of soil, and are expressed as risk/yr per pCi/gram soil. It should be noted that the dose delivered to tissues from external radiation occurs only while the radiation field is present. However, the dose delivered to body tissues due to internal radionuclides consumed in soil, water, and/or food continues long after intake of the radionuclide has ceased

Radionuclide concentrations in air, water or soil are multiplied by intake rates for internal exposure, or by exposure times for external exposure, and then multiplied by USEPA slope factors to estimate potential health risk. Radionuclide intake can also be multiplied by a dose coefficient to estimate equivalent dose, which can then be compared to a radiation protection standard. Differences in the biological effects of different types of ionizing radiation (i.e., alpha, beta, gamma) are accounted for in the dose coefficients. Equivalent dose can be calculated for the whole body when there is uniform irradiation of all tissues, or for individual organs when selected tissues are irradiated non-uniformly. Rem (radiation equivalent man) is the conventional use of dose equivalent. The corresponding SI unit, the Sievert, is equal to 100 rem. Absorbed dose is the energy deposited by ionizing radiation per unit mass of absorbing material (i.e., tissue). Ionizing radiation can only have adverse effects on biological tissues when the radiation is absorbed in tissue. The conventional unit is the rad which is equal to 100 erg per gram. The SI unit, gray, is equal to 100 rad

The slope factors and weight-of-evidence classifications for the radionuclides of concern at OU-2 are presented in Section 3.0



This section presents the toxicity factors for noncarcinogenic and carcinogenic chemical health effects and radiation health effects which are proposed for use in the toxicity assessment of the human health risk assessment for OU-2. It also includes a discussion on the approach proposed for selecting toxicity factors for dermal exposure.

3.1 INHALATION AND ORAL TOXICITY FACTORS

Table 3-1 contains the toxicity factors for noncarcinogenic health effects (RfDs) and for carcinogenic health effects (slope factors) for potential chemicals of concern at OU-2 Toxicity factors for inhalation and ingestion exposure are included the table if available. The sources of the toxicity factors are EPA's 1992 Health Effects Assessment Summary Tables (HEAST) and the Integrated Risk Information System (IRIS). For chemicals where a toxicity factor has been withdrawn by EPA and is under review (i.e., trichloroethene) the 1991 HEAST value, if available, is proposed for use in the risk assessment. The weight-of-evidence for chemicals with carcinogenic toxicity factors is also included in Table 3-1

Table 3-2 contains the texicity factors for carcinogenic health effects of radionuclides of concern due to inhalation, ingestion, and external exposure EPA considers the critical effect of radionuclides to be carcinogenesis and the weight-of-evidence to be Class A, definite evidence of human carcinogenicity.

3.2 DERMAL TOXICITY FACTORS

Oral toxicity factors are generally used to evaluate the toxicity of chemicals absorbed through the skin during dermal contact with contaminated media. This approach is acknowledged by EPA (USEPA 1989). Oral toxicity factors relate the toxic response to an administered (i.e., ingested) dose of chemicals, only some of which may be absorbed by the body, whereas dermal absorption results in an absorbed dose of chemicals. Because of this, EPA (USEPA 1989) suggests adjusting the oral toxicity factors by chemical specific gastrointestinal absorption rates, if available, to yield toxicity factors for dermally absorbed chemicals. Since chemical-specific gastrointestinal absorption rates are not available for most chemicals, this approach has not



TABLE 3-1 TOXICITY FACTORS

A . • .	Oral Slope	Oral RfD	Inhalation Slope	Inhalation RfD	Weight of
Analyte	1/(mg/kg/day)	(mg/kg/day)	l/(mg/kg/day)	(mg/kg/day)	Evidence
1,1,1,2-tetrachloroethane	2 6E-02 (1)	3 00E-02 (1)	2 60E-02 (1)	2 005 01 (2)	С
1,1,1-trichloroethane	2 005 01 (1)	9 00E-02 (2)	2 005 01 (1)	3 00E-01 (2)	•
1,1,2,2-tetrachloroethane	2 00E-01 (1)	4 005 00 (1)	2 00E-01 (1)	•	C
1,1,2-trichloroethane	5 70E-02 (1)	4 00E-03 (1)	5 70E-02 (1)	-	C
1,1-dichloroethane	-	1 00E-01 (3)		1 40E-01 (2)	C
1,1-dichloroethene	6 00E-01 (1)	9 00E-03 (1)	1 75E/01 (1)	-	С
1,2,3-trichloropropane	•	6 00E-03 (1)	- '	-	-
1,2,4-trichlorobenzene	- 407.00 (0)	1 00E-02 (1)	4	3 00E-03 (2)	•
1,2-dibromo-3-chloropropane	1 40E+00 (2)	-	2 40E-03 (2)	5 00E-05 (1)	B2
1,2-dibromoethane	8 50E+01 (1)	-	7/60E-01 (2)	-	B2
1,2-dichlorobenzene	•	9 00E-02 (1)	·	4 00E-02 (2)	•
1,2-dichloroethane	9 10E-02 (1)	· /	9 10E-02 (1)	· -	B 2
1,2-dichloroethene	•	9 00E-03 (2)	-	•	-
1,2-dichloropropane	•	- and	-	1 00E-03 (1)	•
1,2-dimethylbenzene (o-xylene)	-	2 00E+0 (1)	•	-	•
1,3-dimethylbenzene (m-xylene)	•	2,00E+0 (1)	•	-	•
1,4-dichlorobenzene	2 40E-02 (2)		•	2 00E-1 (2)	С
2-butanone	••	6 0E-01 (1)	-	3 0E-01 (1)	
4,4'-DDT	3 40E-01 (1)	5 00E-04 (Î)	3.40E-01 (1)	•	B2
4-methyl-2-pentanone	- /	5 00E-02 (2)	\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\	2 00E-02 (2)	-
acenapthene	- (6 00E-02 (1)	•	-	•
acetone	- ``_ ``	1 00E-01 (1)	7 -	-	-
anthracene	- \	3 00E-01 (1)	•	-	•
antimony	- `	4 00E-04 (1)	•	-	-
Aroclor-1254	7.70E+00 (1)	1 2-	-	-	B2
arsenic	1.75E+00 (1)	3 00E-04 (1)	1 50E+01 (1)	•	A
barium	/ · ·	7 00E/02 (1)	-	1 40E-04 (2)	•
benzene	2 90E-02 (1)		2 90E-02 (2)	-	A
benzo(a)anthracene	5,80E-01 (4)	/-	-	-	B2
benzo(a)pyrene	√5 89É+00 (4)	-	6 10E+00 (2)	- 1	B2
benzo(b)fluoranthene	5 80E-01 (4)	-	•	-	B2
benzo(k)fluoranthene	3-80E-01 (4)	-	•	-	B2
benzoic acid		4 00E+00 (1)	•	-	-
bis(2-ethylhexyl)phthalate	1 40E-02 (1)	2 00E-02 (1)	-	-	B2
bromodichloromethane	6 20E-02 (1)	2 00E-02 (1)	-		B2
bromoform	7.90E-03 (1)	2 00E-02 (1)	3 90E-03 (2)	- 1	B 2
butyl benzylphthalate	, -	2 00E-01 (1)	-	-	-
cadmium (food)		1 0E-03 (1)	6 30E+00 (1)	-	B1
cadmium (water)	-	5 00E-04 (1)	6 30E+00 (1)	-	B 1
carbon tetrachloride	1 30E-01(1)	7 00E-04 (1)	5 25E-02 (1)	-	B2
chlorobenzene	• ` ′	2 00E-02 (1)	•	5 00E-03 (3)	•
chloroethane		•	•	3 00E+00 (1)	-
chloroform	6 10E-03 (1)	1 00E-02 (1)	8 00E-02 (1)	<u>-</u>	B 2
chloromethane	1 30E-02 (2)	(-/	6 30E-03 (2)	<u>.</u>	C
chromium III	-	1 00E+00 (1)	-		•
chrysene	5 80E-02 (4)	-	-	_	B2
cis-1,2-dichloroethene	•	1 00E-02 (2)	-	_	•



TABLE 3-1 TOXICITY FACTORS

	Oral Slope	Oral RfD	Inhalation Slope	Inhalation RfD	Weight of
Analyte	1/(mg/kg/day)	(mg/kg/day)	1/(mg/kg/day)	(mg/kg/day)	Evidence
cis-1,3-dichloropropene	-	3 00E-04 (1)*	•	5 00E-03 (1)*	B2
cumene	-	4 00E-02 (1)	-	3 00E-03 (2)	-
cyanide	-	2 00E-02 (1)	-	-	-
dı-n-butylphthalate	-	1 00E+01 (1)	-	-	-
dı-n-octylphthalate	-	2 00E-02 (2)	- 🧥	-	-
dibromomethane		1 00E-02 (3)	+' }	-	.
dichlorodifluoromethane	-	2 00E-01 (1)	g - (5 00E-02 (3)	-
diethyl phthalate	-	8 00E-01 (1)	.	-	-
ethylbenzene	-	1 00E-01 (1)	/ ^• `\	3 00E-01 (1)	-
fluoranthene	-	4 00E-02 (1)	// - \	-	-
fluorene	-	4 00E-02 (1)	· · ·	· -	•
heptachlor epoxide	9 10E+00 (1)	1 30E-05 (1)	9 10E+00 (1)	\	B2
hexachlorobutadiene	7 80E-02 (1)	-//	7 80E-02 (2)	•	С
hexachloroethane	1 40E-02 (1)	1 00E-63 (1)	1 40E-02 (1)		С
ındeno(1,2,3-cd)pyrene	5 80E-01 (4)	/ - /	•	•	B2
manganese	-	1 90E-Q1 (3)/	-	1 10E-04 (1)	-
mercury	-	3 00E-04 (2)	-	9 0E-05 (2)	-
methylene chloride	7 50E-03 (1)	6 00E-02-(1)	1 60E-03 (1)	9 0E-01 (2)	B2
molybdenum	-	5 00E-03 (1)		-	-
N-nitrosodiphenylamine	4 90E-03 (1)	_	7 -	•	B2
naphthalene	- (4 00E=02-(2)	•	-	-
o-chlorotoluene	- ',	2 00E-02 (1)	7 -	-	-
p-xylene	- `\	2.00E+00 (1)	•	-	-
pentachlorophenol	1 20E-01(1)	3 00E/02 (1)	•	-	B2
pyrene		3,005,02 (1)	-	-	-
silver	/\	5 ÔQE-03 (1)	-	•	-
styrene	/ }	2 00B-Ø1 (1)	•	3 00E-01 (1)	-
tetrachloroethene	5 10E-02 (3)	1-00E-02 (1)	1 80E-03 (3)	-	B2
thallium / <	//	7.00E-05 (2)	•	•	-
toluene	N' /-	2 00E-01 (1)	-	1 10E-01 (1)	-
trans-1,2-dichloroethene	1	2 00E-02 (1)	-	•	-
trichloroethene	1 10E-02-(3)	•	5 95E-03 (3)	•	B2
vinyl chloride	1 90E+Q (1)	<u> </u>	3 00E-01 (1)	•	Α

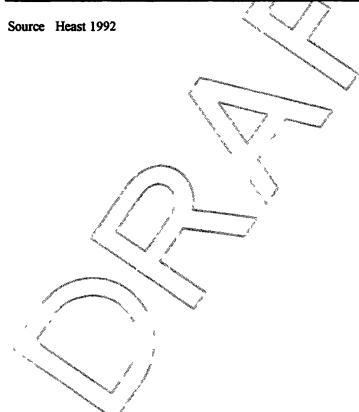
Sources

- 1 = IRIS
- 2 = HEAST 1992
- 3 = HEAST 1991
- 4 = EPA Region IV Guidance, February 1992
- * Values are for 1,3-dichloropropene No data for individual isomer
- Not classifiable or not carcinogenic or No toxicity value available



TABLE 3-2 TOXICITY FACTORS FOR RADIONUCLIDES

Analyte	Oral Slope Factor (Rısk/pCı)	Inhalation Slope Factor (Risk/pCi)	External Slope Factor (Risk/yr/pCi/g)	Weight of Evidence
241 Americium	2 4E-10	3 2E-08	4 9E -09	Α
134 Cesium	4 1E-11	2 8E-11	€ \$ 2E-06	Α
137 Cesium*	2 8E-11	1 9E-11	/ /0 0E+00	Α
238 Plutonium	2 2E-10	3 9E-08	< 2 8E-11	A
239 Plutonium	2 3E-10	3 8E-08 🥖	1.7E-11	A
240 Plutonium	2 3E-10	3 8E-08 / /	2 7E-11	A
226 Radium*	1 2E-10	3 0E-09√√	1 2E-08	A
228 Radium*	1 0E-10	6 6₹-10	0 0E+00	A
Strontium 89	3 0E-12	2,9E-₹2	4 7E-10	Α
Strontium 90	3 3E-11	∕5 6Æ-11	0 0E+00	A
Tritium	5 4E-14	/ 7/8E-14	0 0E+00	Α





been adopted in this toxicity assessment. If dermal absorption of particular chemicals is demonstrated to be a potential significant contributor to overall risk in the risk assessment, a more detailed analysis of the toxicity factors may be warranted

3.3 DERMAL PERMEABILITY CONSTANTS AND ABSORPTION FACTORS

Dermal permeability constants for organic chemical in aqueous solution are used to estimate the amount of chemical absorbed from surface water or sediment that may be contaminated by migration of chemicals of concern from groundwater or surface soils.

Dermal permeability constants for aqueous solutions are presented in EPA's Dermal Exposure Assessment guidance (January 1992) and are proposed for use in the risk assessment (USEPA 1992b) Dermal exposure to inorganic chemicals of concern (i.e., metals and radionuclides) in groundwater that may migrate to surface water and sediments will be evaluated qualitatively in the risk assessment

Table 3-3 contains the dermal permeability constants proposed for aqueous solutions of organic chemicals of concern in groundwater that may migrate to surface water and sediments. No organic chemicals of concern were identified for surface soils in the Draft Chemicals of Concern Technical Memorandum No 9 for OU-2 (July 1993)

The Chemicals of Concern Technical Memorandum states that the dermal absorbed fraction of organics in subsurface and surface soils will be assumed to be 10 percent. The following section provides the rationale for this assumption

The absorbed fraction in the intake factor equation for dermal absorption is the estimated fraction of organic compounds adhered to soil particles that partitions to and is absorbed through skin. Percent absorbed depends upon soil loading, organic carbon content of soil, contaminant concentration, duration of exposure, animal species used in the experiment, and whether the experiment is conducted in vitro or in vivo. For purposes of this risk assessment, an upperbound estimate of absorption rate for organic compounds adhered to soil particles is assumed to be 10 percent. These rates are based on experimental results using B(a)P in acetone or in crude oil, and adjusting the absorption rates for shorter exposure duration and



TABLE 3-3 DERMAL PERMEABILITY CONSTANTS FOR GROUNDWATER CHEMICALS OF CONCERN

Chemical	Kp (cm/hr)
1,2-dibromoethane	-
1,1-dichlorethene	1 60E-02
cis-1,2-dichloroethene	1 00E_02
carbon tetrachloride	2 20É-02
chloroform	8,90E-03
methylene chloride	A 50E-03
tetrachloroethene	4.80E-02
trichloroethene	1 60E-02
vinyl chloride	7 30E-03

Source

Permeability constants taken from Dermal Exposure Assessment Principles and Applications, Table 5-7 (EPA 1992)

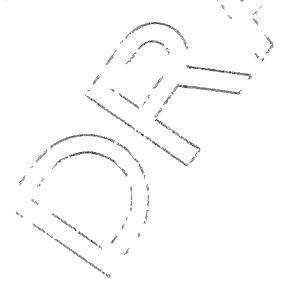
- not available



the observed retarding effect of the soil medium¹ The experimental results are summarized in Table 3-4, Percent Dermal Absorption of Neat Benzo(a)pyrene at 24 hours. Absorption rates range from 3 to 51 percent at 24 hours. The arithmetic mean absorption rate is 17 percent, and the 95 percent. UCL on the mean rate is 26 percent. To adjust these experimental rates to account for site-specific exposure conditions, it is assumed that the exposed individual showers within 12 hours of exposure, and that absorption from soil is one-fifth that of the pure compound (Yang et al. 1989, Wester et al. 1990). Therefore, the 24-hour absorption rates of heat B(a)P are adjusted by a factor of 0.5 for a 12-hour exposure and 0.2 for the soil matrix effect. Resulting absorption rates are

 $26 \times 05 \times 02 = 26$ percent

It should be noted that B(a)P is one of the more lipophilic of the PAHs, and therefore it may be absorbed at a higher rate than a number of other organic chemicals of concern. Also, the use of dermal absorption values obtained in experimental animal studies will almost always result in a conservative (i.e., higher) estimate of dermal absorbed dose in humans (EPA 1992b). Therefore, the dermal absorption rate used in this analysis (10 percent) is concluded to be a conservative estimate of a reasonable maximum rate of dermal absorption of organic compounds from soil



In recent guidance on dermal exposure assessments (EPA 1992a), EPA has declined to recommend an absorption rate for B(a)P is soil because of the variability in experimental conditions and results and the difficulty in extrapolating from high soil loadings (e.g., tens of mg/cm²) under experimental conditions to lower loading (e.g., 1 mg/cm²) typical of human exposures (EPA 1992a) (B(a)P at concentrations of 1 and 10 mg/kg and soil loadings of 40 to 56 mg/cm², experimental results for percent absorbed at 24 hours ranges from 1 percent [Yang et al 1989] to 13 percent [Wester et al 1990])

TABLE 3-4 PERCENT DERMAL ABSORPTION OF BENZO(a)PYRENE AT 24 HOURS

9/4	Re	P	he	orbe	d

	,	, u		
Source(1)	at 24 hour	Preparation	Vehicle	Dose
Yange et al 1986	6	Rat in vivo	Acetone	9-10 ug/cm^2
	17	Rat in vitro	Acetone	9-10 ug/cm^2
Yang et al 1989	6	Rat in vivo	Appm BaP in crude oil	90 ug/cm^2
	12	Rat in vitro	1 ppm Bal un crude oil	90 ug/cm^2
Kao et al 1984	24	Mouse in vitro	Acetone	1 ug/cm^2
Kao et al 1985	3	Human in vitro	Acetone	2 ug/cm^2
Kao et al 1988	10	Mice in vitro	Acetone	2 5 ug/cm^2
Wester 1990	24	Human in vitre	Acetone	10 ppm
	51	Rhesus monkey in vivo	Acetone	10 ppm
Average % absorbed	17			
95% UCL % Absorbed	25 68			

Kao et al 1984 Toxicology and Applied Pharmacology 75-289-298

Kao et al 1985 Toxicology and Applied Pharmacology-81.502-516

Kao et al 1986 Toxicology and Applied Pharmacology 94 93 103

Yang et al 1986 Toxicology and Industrial Health 2 409-416

Yang et al 1989 Bulletin of Environmental Contaminants and Toxicology 43 207-214

Wester et al 1990 Fundamentals of Applied Toxicology 15 \$10-516

(1) The cited studies are from the references cited in EPA 1992 Dermal Exposure Assessment Principles and Applications (EPA/800/8-91/011B) Studies not cited in this table include those conducted in previously frozen tissue and Sanders et al. 1984 (in vivo percutaneous absorption of BaP in mouse)

The latter was excluded because mouse skin has been shown to be 2.5 to 5 times more permeable than skin of other species, including human (Kao et al. 1985, as cited in EPA 1992 Dermal Exposure Assessment Principles and Applications).



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